

Attachment B

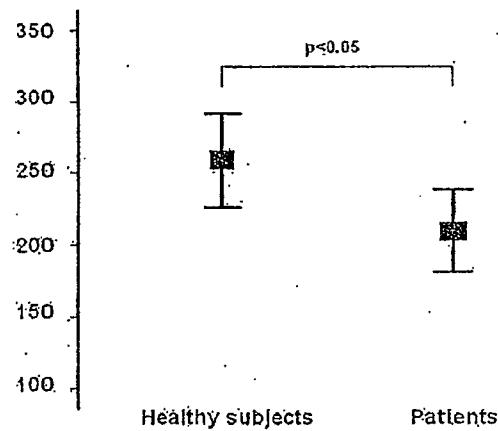
Experimental data for an EPO therapy with patients having a dysfunction of endothelial progenitor cells, cardiovascular risk factors and end-organ damages:

Patients with cardiovascular risk factors and end-organ damages show a dysfunction of their endothelial progenitor cells.

Figure 1 shows a quantitative determination of cultivated endothelial progenitor cells (EPC). The figure shows that the absolute number of endothelial progenitor cells in 38 hypertonic type II diabetic patients is significantly lower compared to 38 age- and sex-matched control persons. The data is depicted as 95% confidence interval of the mean value.

Figur 1:

EPCs (per high power-field)

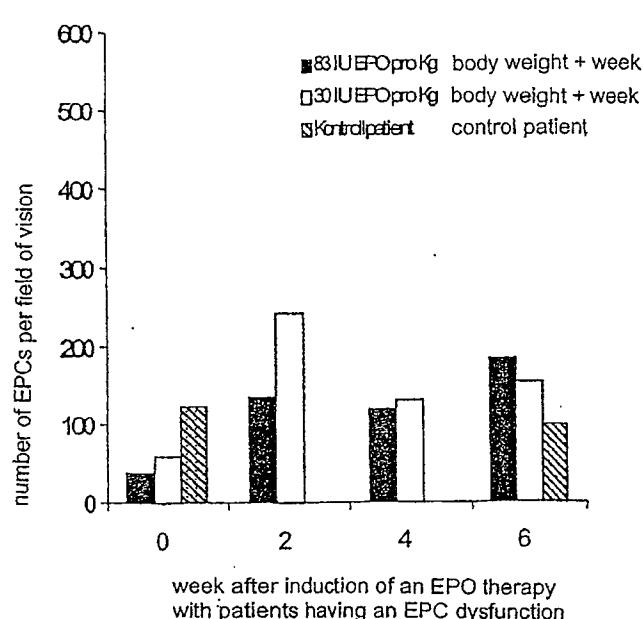


The application of EPO abolishes the EPC dysfunction.

Figure 2a shows a quantitative determination of cultivated endothelial progenitor cells (EPC) of two hypertonic patients with already decreased kidney function, wherein the two patients were treated with an EPO therapy according to the present invention, and further of a control patient without EPO therapy. Figure 2b shows a quantitative determination of cultivated endothelial progenitor cells (EPC) from 46 healthy subjects.

The figures show that the patients were suffering from an EPC dysfunction. The number of the EPCs quantified with the present assay is significantly reduced in comparison to the control collective of 46 healthy subjects. Already after two weeks after the beginning of an EPO therapy the EPC dysfunction could be abolished. The number of EPCs was almost the same compared to the value of the healthy control group. The EPO therapy could be successfully conducted with a weekly dosage of only 83 IU/kg body weight and only a weekly application of only 30 IU/kg body weight. However, one patient, excluded from the EPO therapy as control, showed a diminished EPC dysfunction over a time period of six weeks.

Figur 2a:



Figur 2b:

